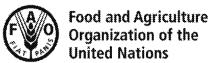
UNITED NATIONS









UNEP/FAO/RC/COP.9/10/Add.1



Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade

Distr.: General 29 October 2018 Original: English

Conference of the Parties to the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade Ninth meeting
Geneva, 29 April–10 May 2019
Item 5 (b) of the provisional agenda*

Metters related to the implementation of the

Matters related to the implementation of the Convention: listing of chemicals in Annex III to the Convention

Inclusion of chrysotile asbestos in Annex III to the Rotterdam Convention

Addendum

Draft decision guidance document

Note by the Secretariat

As is mentioned in document UNEP/FAO/RC/COP.9/10, the Chemical Review Committee at its second meeting agreed on the text of a draft decision guidance document on chrysotile asbestos. ¹ The draft decision guidance document is set out in the annex to the present note for the consideration of the Conference of the Parties. It has not been formally edited.

^{*} UNEP/FAO/RC/COP.9/1.

¹ UNEP/FAO/RC/CRC.2/20, para. 106 and annex I.

Annex

Rotterdam Convention – Operation of the Prior Informed Consent procedure for banned or severely restricted chemicals

Draft Decision Guidance Document

Chrysotile Asbestos







Food and Agriculture Organization of the United Nations

Secretariat of the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade



Introduction

The objective of the Rotterdam Convention is to promote shared responsibility and cooperative efforts among Parties in the international trade of certain hazardous chemicals in order to protect human health and the environment from potential harm and to contribute to their environmentally sound use, by facilitating information exchange about their characteristics by providing for a national decision-making process on their import and export and by disseminating these decisions to Parties. The secretariat of the Convention is provided jointly by the United Nations Environment Programme (UNEP) and the Food and Agriculture Organization of the United Nations (FAO).

Candidate chemicals for the Rotterdam Convention include those that have been banned or severely restricted by national regulatory actions in two or more Parties¹ in two different regions. Inclusion of a chemical in the Convention is based on regulatory actions taken by Parties that have addressed the risks associated with the chemical by banning or severely restricting it. Other ways might be available to control/reduce such risks. However, inclusion does not imply that all Parties to the Convention have banned or severely restricted this chemical. For each chemical included in the Rotterdam Convention, Parties are requested to make an informed decision whether they consent or not to the future import of the chemical.

At its XXXX meeting, held in XXXX on XXXX the Conference of the Parties adopted the decision guidance document for chrysotile asbestos with the effect that this chemical became subject to the PIC procedure.

The present decision guidance document was communicated to the Designated National Authorities on [xxxx] in accordance with Articles 7 and 10 of the Rotterdam Convention.

Purpose of the Decision Guidance Document

For each chemical included in the PIC procedure a decision guidance document has been approved by the Conference of the Parties. Decision guidance documents are sent to all Parties with a request that they provide a decision regarding future import of the chemical.

The decision guidance document is prepared by the Chemical Review Committee (CRC). The CRC is a group of government designated experts established in line with Article 18 of the Convention that evaluates candidate chemicals for possible inclusion in the Convention. The decision guidance document reflects the information provided by two or more Parties in support of the national regulatory actions to ban or severely restrict the chemical. It is not intended as the only source of information on a chemical nor is it updated or revised following its adoption by the Conference of the Parties.

There may be additional Parties that have taken regulatory actions to ban or severely restrict the chemical as well as others that have not banned or severely restricted it. Such risk evaluations or information on alternative risk mitigation measures submitted by Parties may be found on the Rotterdam Convention web-site (www.pic.int).

Under Article 14 of the Convention, Parties can exchange scientific, technical, economic and legal information concerning the chemicals under the scope of the Convention including toxicological, ecotoxicological and safety information. This information may be provided directly to other Parties or through the Secretariat. Information provided to the Secretariat will be posted on the Rotterdam Convention website.

Information on the chemical may also be available from other sources.

¹ "Party' means a State or regional economic integration organization that has consented to be bound by this Convention and for which the Convention is in force."

Disclaimer

The use of trade names in this document is primarily intended to facilitate the correct identification of the chemical. It is not intended to imply any approval or disapproval of any particular company. As it is not possible to include all trade names presently in use, only a number of commonly used and published trade names have been included in this document.

While the information provided is believed to be accurate according to data available at the time of preparation of this Decision Guidance Document, the Food and Agriculture Organization of the United Nations (FAO) and the United Nations Environment Programme (UNEP) disclaim any responsibility for omissions or any consequences that may flow there from. Neither FAO nor UNEP shall be liable for any injury, loss, damage or prejudice of any kind that may be suffered as a result of importing or prohibiting the import of this chemical.

The designations employed and the presentation of material in this publication do not imply the expression of any opinion whatsoever on the part of FAO or UNEP concerning the legal status of any country, territory, city or area or of its authorities or concerning the delimitation of its frontiers or boundaries.

j i	ABBREVIATIONS WHICH MAY BE USED IN THIS DOCUMENT		
(N.B. Chem	nical elements and pesticides are not included in this list)		
<	less than		
<	less than or equal to		
<<	much less than		
>	greater than		
>	greater than or equal to		
μg	Microgram		
μm	Micrometre		
Paris			
a.i.	active ingredient		
ACGIH	American Conference of Governmental Industrial Hygienists		
ADI	acceptable daily intake		
ADP	adenosine diphosphate		
ATP	adenosine triphosphate		
b.p.	boiling point		
bw	body weight		
oC	degree Celsius (centigrade)		
CA	Chemicals Association		
CAF	Compressed asbestos fibre		
cc	Cubic centimetre		
CCPR	Codex Committee on Pesticide Residues		
СНО	Chinese hamster ovary		
cm	centimetre		
CSTEE	E.C. Scientific Committee on Toxicity, Ecotoxicity and the Environment		
00122	2.07 Services Committee of Tollier, 2.00 man in 2.00 miles		
D	Dust		
DNA	Deoxyribose Nucleic Acid		
	-,		
E.C.	European Community		
EC50	Effect concentration, 50%		
ED50	Effect dose, 50%		
EEC	European Economic Community		
EHC	Environmental Health Criteria		
ERL	Extraneous residue limit		
FAO	Food and Agriculture Organization of the United Nations		
g	Gram		
GL	Guideline level		
GR	Granules		
h	Hour		
ha	Hectare		
i.m.	Intramuscular		
i.p.	Intraperitoneal		
IARC	International Agency for Research on Cancer		
IC50	Inhibition concentration, 50%;		
ILO	International Labour Organisation		
IPCS	International Programme on Chemical Safety		
IRPTC	International Register of Potentially Toxic Chemicals		
IUPAC	International Union of Pure and Applied Chemistry		
JMPR	Joint FAO/WHO Meeting on Pesticide Residues (Joint Meeting of the FAO Panel of Experts		
	on Pesticide Residues in Food and the Environment and a WHO Expert Group on Pesticide		
	Residues)		
k	Kilo- (x 1000)		
kg	Kilogram		
Koc	Organic carbon-water partition coefficient		
1	Litre		
LC50	Lethal concentration, 50%		
LD50	Lethal dose, 50%		
LOAEL	Lowest observed adverse effect level		
LDLO	Lowest lethal dose		

ABBREVIATIONS WHICH MAY BE USED IN THIS DOCUMENT

(N.B. Chemical elements and pesticides are not included in this list)

LOEL lowest observed effect level

m Metre
m.p. melting point
mg Milligram
ml Millilitre
mPa MilliPascal

MRL maximum residue limit MTD maximum tolerated dose

NCI National Cancer Institute (United States)

ng Nanogram

NIOSH National Institute of Occupational Safety and Health (United States)

NOAEL no-observed-adverse-effect level

NOEL no-observed-effect level

NOHSC National Occupational Health and Safety Commission (Australia)

NTP National Toxicology Program

OECD Organisation for Economic Co-operation and Development

OP organophosphorus pesticide

PCM Phase contrast microscopy PHI pre-harvest interval PIC Prior Informed Consent

Pow octanol-water partition coefficient POP persistent organic pollutant

ppm parts per million (used only with reference to the concentration of a pesticide in an

experimental diet. In all other contexts the terms mg/kg or mg/l are used).

RfD reference dose for chronic oral exposure (comparable to ADI)

SBC secretariat for the Basel Convention

SC Soluble concentrate
SG water soluble granules
SL soluble concentrate
SMR standardized mortality ratio
STEL short term exposure limit

TADI temporary acceptable daily intake

TLV threshold limit value

TMDI theoretical maximum daily intake TMRL temporary maximum residue limit

TWA time weighted average

UNEP United Nations Environment Programme
USEPA United States Environmental Protection Agency

UV Ultraviolet

VOC volatile organic compound

WHO World Health Organization

WP wettable powder wt Weight

CHRYSOTILE ASBESTOS

1. Identification and uses (see Annex 1) - Chrysotile

Common name Chrysotile

Chemical name Chrysotile Asbestos

Other names/synonyms Asbestos, Serpentine asbestos, white asbestos

CAS-No.(s) 12001-29-5

Other CAS numbers that General CAS number for asbestos: 1332–21–4

may be used Additional CAS number for chrysotile 132207-32-0

Harmonized System

Customs Code 2524.00 (asbestos)

Other numbers:

E.C. Number – 650–013–00–6

RTECS number - CI6478500

Molecular formula Mg₃(Si₂O₅)(OH)₄

Structural formula OH

Category Industrial Regulated Category Industrial

Use(s) in regulated category Chrysotile is by far the predominant asbestos fibre consumed today

(94% of the world's production) and is processed into products such as friction materials, asbestos-cement, cement pipe and sheet, gaskets

and seals, paper and textiles (IPCS, 1998). The

asbestos-cement industry is by far the largest user of chrysotile fibres,

accounting for about 85% of all use.

Australia: Chrysotile is also used in blades in high vacuum pumps, asbestos yarn for packing, asbestos gloves and asbestos washers.

European Community: chrysotile diaphragms (see below),

chrysotile-containing spare parts for maintenance.

Trade names 7-45 Asbestos, Avibest, Avibest C, Calidria RG 100, Calidria RG

144, Calidria RG 600, Cassiar AK, K 6-30, NCI C61223A & 5RO4.

This list is an indicative list of trade names and is not intended to be

exhaustive.

Formulation types Chrysotile has been used in the manufacture of a wide range of

articles. Available in solid formulations for the manufacture of

friction materials and gasket production.

Uses in other categories No reported uses as a pesticide chemical.

Basic manufacturers Naturally occurring, mined.

2. Reasons for inclusion in the PIC procedure - Chrysotile

Chrysotile (serpentine forms of asbestos) is included in the PIC procedure as an industrial chemical. It is listed on the basis of the final regulatory actions to ban or severely restrict its use as notified by Australia, Chile and the European Community (EC).

2.1 Final regulatory action: (see Annex 2 for details)

Australia

From 31 December 2003, all new uses of chrysotile asbestos and goods containing chrysotile asbestos were banned in Australia, including the replacement of chrysotile asbestos products when replacement is necessary. It is illegal under the laws of each state and territory to store, sell, install or use any products containing chrysotile asbestos. There are a few exemptions to the ban but these are restricted in scope and operate for a limited time.

Reason: Human Health

Chile

Severely restricted:

Production, importation, distribution, sale and use of construction materials containing any type of asbestos is prohibited.

Production, importation, distribution, sale and use of chrysotile and any other type of asbestos, or mixture thereof, for any item, component or product that does not constitute a construction material is prohibited, with certain specific exceptions. (No exceptions apply to crocidolite.)

Reason: Human Health

European Community

Banned – The placing on the market and use of all forms of asbestos, including chrysotile, and products containing these fibres added intentionally, is prohibited, with one limited exception in the case of chrysotile.

Reason: Human Health

2.2 Risk evaluation

Australia

The National Industrial Chemicals Notification and Assessment Scheme (NICNAS) undertook a risk assessment of chrysotile in 1995 and published the final report in February 1999. It assessed the occupational, public health and environmental risks associated with uses and applications in Australian industry. It also assessed the feasibility of substitution of chrysotile materials and voluntary and/or legislative action for reducing potential health and safety risks arising from manufacture and import of chrysotile products. The risk assessment concluded that human exposure to chrysotile is associated with an excess risk of asbestosis, lung cancer and mesothelioma. However, there are many confounding factors surrounding risk estimates for chrysotile exposure, such as the possibility of a threshold effect, possible co-exposure to other fibre types, inaccurate estimates of historical exposures and the influence of tobacco smoking.

Chile

A hazard evaluation was carried out based on a compilation of bibliographic sources and verification of adverse chronic effects in exposed workers in the asbestos cement industry. It was concluded that those at greatest risk are workers who handle asbestos fibres for various uses. In Chile, this means in particular those workers who have been exposed to fibres from the manufacture of construction materials.

European Community

An independent risk assessment was undertaken. This confirmed that all forms of asbestos can cause lung cancer, mesothelioma, and asbestosis; and that no threshold level of exposure could be identified below which asbestos does not pose carcinogenic risks.

3. Protective measures that have been applied concerning the chemical – Chrysotile

3.1 Regulatory measures to reduce exposure

Australia

Protective measures were taken by prohibiting all new uses of chrysotile asbestos and goods containing chrysotile asbestos, including the replacement of chrysotile asbestos products when replacement is necessary. There are a few exemptions to the ban but these are restricted in scope and operate for a limited time (see Annex 2 for further details).

Chile

Protective measures were taken by prohibiting all uses of all types of asbestos for use as an input to the manufacture of construction materials.

All types of asbestos prohibited for use for any item, component or product that does not constitute a construction material unless excepted.

Any type of asbestos (except crocidolite): the use of asbestos may be authorized in the manufacture of products or components that are not construction materials so long as the interested parties can prove that there is no technically or economically feasible substitute for it.

European Community

Protective measures were taken by prohibiting the placing on the market and use of chrysotile and of products containing these fibres added intentionally, with one specific exception in respect of diaphragms for existing electrolysis installations (see Annex 2 for further details).

3.2 Other measures to reduce exposure

Australia

Code of Practice for the Management and Control of Asbestos in Workplaces [NOHSC: 2018(2004)] Guidance provided in documents available from NOHSC website at

http://www.nohsc.gov.au/OHSLegalObligations/NationalStandards/asbest.htm are:

Code of Practice for the Safe Removal of Asbestos [NOHSC: 2002 (1988)]

Guidance Note on the Membrane Filter Method for Estimating Airborne Asbestos Dust [NOHSC: 3003(1988)]

Guide to the Control of Asbestos Hazards in Buildings and Structures [NOHSC: 2002 (1998)]

European Community

Directive on the demolition of buildings, structures and installations containing asbestos and the removal of asbestos or materials containing asbestos therefrom (Council Directive 87/217/EEC (OJ L 85, 28.3.1987, p.40), as amended by Council Directive 91/692/EEC (OJ L 377, 31.12.1991, p.48)) Directive on disposal of construction materials (Council Directive 91/689/EEC (OJ L 377, 31.12.1991, p.20))

General Dust control by wetting material, use of respirators, use of full protective clothing with attention when further treating any contaminated clothing (information from crocidolite DGD). Further guidance is provided in the ILO Convention No. 162 "Safety in the Use of Asbestos" (http://www.ilo.org/ilolex/cgi-lex/convde.pl?C162) which applies to all activities involving exposure of workers to asbestos in the course of work.

The ILO recommendation 172 (http://www.ilo.org/ilolex/cgi-lex/convde.pl?R172), contains recommendations on safety in the use of asbestos, including details on protective and preventative measures, surveillance of the working environment and workers' health, information and education measures.

More specific information on measures to reduce exposures on construction sites is provided in the International Standard Organisation (ISO) 7337 "Asbestos-reinforced cement products – Guidelines for on-site work practices."

3.3 Alternatives

It is essential that before a country considers substituting alternatives, it ensures that the use is relevant to its national needs, and the anticipated local conditions of use. The hazards of the substitute materials and the controls needed for safe use should also be evaluated.

Australia

Alternatives have been developed for most uses of chrysotile in Australia. A discussion of chrysotile alternatives is provided in the NICNAS Priority Existing Chemical report on Chrysotile Asbestos. This report can be accessed from:

http://www.nicnas.gov.au/publications/CAR/PEC/PEC9/PEC9index.asp

Chile

It has been proved that it is feasible to replace asbestos with other fibres in manufacturing fibro-cement materials and still obtain products of similar quality. In fact, the company producing the greatest quantity of panels and sheeting for dwellings in Chile has replaced asbestos with other fibres such as cellulose. In the case of brake parts, asbestos-containing and asbestos-free brake pads and linings are in use, until the existing in use asbestos-containing brake pads and linings at the time of publication of the prohibition should be replaced.

European Community

Identified alternatives include cellulose fibres, polyvinyl alcohol (PVA) fibres and P-aramid fibres.

General

Guidance on substituting alternatives to chrysotile asbestos fibres is provided in IPCS Environmental Health Criteria 151 "Selected Synthetic Organic Fibres".

3.4 Socio-economic effects

Countries should consider the results of this information in the context of their own national conditions.

Australia

The National Occupational Health and Safety Commission (NOHSC) commissioned a report on the economic impact of prohibition in March 2001. The report recommended that a legislative ban on importation and use of chrysotile products within Australia be introduced over a five-year period. A ban on the use of chrysotile is expected to have significant benefits through a reduction in illness and deaths from future exposure to the material. This would translate to a reduction in costs to the community. However, there would be costs incurred by large and small businesses initially due to projected higher costs of chrysotile asbestos substitutes.

Chile

No assessment of socio-economic effects was undertaken.

European Community

The prohibition in respect of chrysotile had to be implemented at the latest by 1st January 2005, but Member States were able to implement it as from 26.8.1999. A study into the economic implications of replacing asbestos cement products and the availability of alternatives concluded that about 1500 jobs would be lost in some Member States of the European Community and that there could be subsequently rather severe effects on local economics in the regions concerned. However, the impact would be softened, if a 5-year transitional period was foreseen, and through the creation of new jobs in other sectors.

4. Hazards and risks to human health and/or the environment - Chrysotile

4.1 Hazard Classification		
IARC Carcinogenic to humans (<i>Group 1</i>) IARC (1987)		
European	Carc. Cat. 1	
Community	R45 May cause cancer	
-	T:R48/23 Toxic: danger of serious damage to health by prolonged exposure	
	through inhalation (E.C., 2001)	
NTP	Chrysotile is classified as "Known Human Carcinogen" (US, 2001)	

4.2 Exposure limits

No internationally agreed exposure limits available

4.3 Packaging and labelling

The United Nations Committee of Experts on the Transportation of Dangerous Goods classifies the chemical in:

Hazard Class	UN number 2590			
and Packing	Class 9 – Miscellaneous dangerous goods and articles			
Group	Proper shipping name: WHITE ASBESTOS			
	Packaging group:	III		
	Emergency Procedure Guide:	9B7		
	Special Provision number:	168		
	Packaging method:	3,8.9		
	General: Mineral fibres of varying length. Non-combustible. Inhalation of the dust			
	of asbestos fibres is dangerous and therefore exposure to the dust should be			
	avoided at all times. Always prevent the generation of asbestos dust. A safe level			
	of airborne concentration of asbestos fibres may be obtained through effective			
	packaging or unitizing. Compartments and vehicles or containers that have			
	contained asbestos should be carefully cleaned before receiving other cargo.			
	Hosing down or vacuum cleaning as appropriate, instead of sweeping will prevent the atmosphere from becoming dust laden. This entry may also include talc			
	containing tremolite and/or actinolite.	en. This entry may also include tale		
International				
Maritime	UN No: 2590: Class or division 9			
Dangerous				
Goods				
(IMDG) Code				
Transport	TEC (R) -913			
Emergency	1 LC (R) ->13			
Card				

4.4 First aid

NOTE: The following advice was correct at the time of publication. This advice is provided for information only and is not intended to supersede any national first aid protocols.

Not acutely toxic. In case of exposure, prevent dispersion of dust. Avoid all contact. Avoid exposure of adolescents and children. There is no antidote. Seek medical advice.

4.5 Waste management

Chrysotile asbestos may be recovered from waste slurries. Otherwise friable waste should be wetted and containerised (sealed, double bagging) to avoid dust formation during transport and disposal. Landfilling is recommended in a supervised landfill and, waste should initially be covered with at least 15 cm of soil. For final closure of an area containing asbestos a cover of at least 1 m of compacted soil should be applied.

Annexes	
Annex 1	Further information on the substance
Annex 2	Details on Final regulatory action
Annex 3	Address of designated national authorities
Annex 4	References

Introduction to Annex I

The information presented in this Annex reflects the conclusions of the notifying parties: Australia, Chile and European Community. In a general way, information provided by these parties on these hazards are synthesised and presented together, while the risk assessments, specific to the conditions prevailing in these countries, are presented separately. This information is contained in the documents referenced in the notifications in support of their final regulatory actions banning chrysotile asbestos, including international reviews. The notification from Australia was first reported in PIC Circular XIX of June 2004, the notification from Chile was first reported in the PIC Circular XV of June 2002 and the notification from the European Community in PIC Circular XIII of June 2001.

Chrysotile asbestos was included as a subject of an IPCS Environmental Health Criteria document (Asbestos and other Natural Mineral Fibres, EHC 53) published in 1986. It was also reviewed in the IPCS Environmental Health Criteria Document (Chrysotile Asbestos, EHC 203) published in 1998.

Annex 1 - Further information - Chrysotile

1. Physico-Chemical properties

1.1	Identity	Chrysotile
1.2	Formula	$Mg_3(Si_2O_5)(OH)_4$

- 1.3 Colour and Texture Usually white to pale green, yellow, pink. Usually flexible, silky and tough
- **1.4 Decomposition** 450–700°C => 800 850°C temperature
- 1.5 Fusion temperature 1500°C of Residual material
- **1.6 Density** 2.55 g/cm³
- 1.7 Resistance to acids Undergoes fairly rapid attack when compared to amphiboles
- 1.8 Resistance to alkalis Very good
- 1.9 Tensile strength $31 (10^3 \text{ kg/cm}^2)$
- 2 Toxicological properties

2.1 General

Chrysotile is the serpentine form of asbestos. Other variants of asbestos (crocidolite, amosite, actinolite, anthophyllite and tremolite) are amphibole forms

There is general consensus amongst the scientific community that all types of asbestos fibres are carcinogenic (Royal Society of Canada, 1996 cited by E.C., 1997) and can cause asbestosis, lung cancer and mesothelioma when inhaled.

Chrysotile is classified as a known human carcinogen (IARC, 1987). Exposure poses increased risks for asbestosis, lung cancer and mesothelioma in a dose-dependent manner (IPCS, 1998). It has been shown that smoking and asbestos act in a synergistic manner, increasing the overall risk of lung cancer.

In 1998, the EC Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) concluded that chrysotile is a proven carcinogen and there is not sufficient evidence that it acts through a non-genotoxic mechanism (CSTEE 1998).

2.2 Deposition and clearance

Depending largely on size and shape, deposition of inhaled chrysotile asbestos fibres may occur in lung tissue. Some fibres may be removed by mucociliary clearance or macrophages while others may be retained in the lungs for extended periods. Inhalation exposure is, therefore, generally regarded as cumulative, and exposures have been expressed in terms of concentration of fibres over time or PCM fibre-years/ml.

Analyses of human lungs of workers exposed to chrysotile asbestos indicate much greater retention of tremolite, an amphibole asbestos commonly associated with commercial chrysotile in small proportions, than of chrysotile. The more rapid removal of chrysotile fibres from the human lung is further supported by findings from animal studies showing that chrysotile is more rapidly cleared from the lung than are amphiboles including crocidolite and amosite (IPCS, 1998).

2.3 Mode of action

The ability of fibres to induce fibrogenic and carcinogenic effects appears to be dependent on their individual characteristics, including dimension and durability (i.e. biopersistence) in target tissues, which are determined in part by the physico-chemical properties. It is well documented from experimental studies that fibres shorter than 5 µm are less biologically active than fibres longer than 5 µm. However, it is still uncertain whether short fibres have any significant biological activity. Furthermore it is still uncertain as to how long a fibre needs to remain in the lung in order to induce preneoplastic effects (IPCS, 1998).

IPCS (1998) concluded that the significance of physical and chemical properties (e.g. fibre dimension, surface properties) of fibres and their biopersistence in the lung in relation to their biological and pathogenic effects needs further elucidation.

2.4 Effects on experimental animals

Results from animal studies reflect the known human health effects of chrysotile asbestos. IARC (1987) reports that chrysotile produced mesothelioma and lung carcinomas in rats after inhalation and mesothelioma following intrapleural administration. Chrysotile induced mesothelioma in hamsters following intrapleural administration, and peritoneal mesothelioma in mice and rats following intraperitoneal administration. Results of experiments in which chrysotile was given orally to rats or hamsters have been equivocal. For most of these experiments, it is not known whether and to what extent the chrysotile was contaminated with amphiboles (IARC, 1987 cited by CSTEE, 1998). Since the publication of Environmental Health Criteria 53 (IPCS, 1986), there have been only a few studies in which possible harmful effects of the ingestion of chrysotile asbestos have been examined in experimental animals. All these studies gave negative findings.

Various experimental samples of chrysotile fibres have been shown in numerous long-term inhalation studies to cause fibrogenic and carcinogenic effects in laboratory rats. These effects include interstitial fibrosis and cancer in the lung and pleura (Wagner et al, 1974; Wagner et al, 1984; Le Bouffant et al, 1987; Davis et al, 1986; Davis et al, 1988, Bunn et al, 1993, all cited IPCS, 1998). In most cases, there appears to be an association between fibrosis and tumours in the rat lung. These studies using experimental samples of chrysotile such as chrysotile B from the Union International Contre le Cancer (UICC) provide clear evidence that chrysotile in its uncontaminated form causes asbestosis, mesothelioma and lung cancer in animals. Fibrogenic and carcinogenic effects have also been found in long-term animal studies using other modes of administration (e.g. intratracheal instillation and intrapleural or intraperitoneal injection) (Lemaire, 1985, 1991; Lemaire et al, 1985, 1989; Bissonnette et al 1989; Begin et al, 1987 and Sebastien et al, 1990, all cited IPCS, 1998).

Exposure/dose-response relationships for chrysotile-induced pulmonary fibrosis, lung cancer and mesothelioma have not been adequately investigated in long-term animal inhalation studies (IPCS, 1998). Inhalation studies conducted to date, mainly using a single exposure concentration, show fibrogenic and carcinogenic responses at airborne fibre concentrations ranging from 100 to a few thousand fibres/ml. When data from various studies are combined, there appears to be a relationship between airborne fibre concentrations and lung cancer incidence. This type of analysis, however, may not be scientifically sound as different experimental conditions were used in available studies.

In non-inhalation experiments (intrapleural and intraperitoneal injection studies), dose-response relationships for mesothelioma have been demonstrated for chrysotile fibres. However data from these studies may not be suitable for the evaluations of human risk inhalation exposure to fibres (Coffin et al, 1992; Fasske, 1988; Davis et al, 1986, all cited IPCS, 1998).

Overall, the available toxicological data provide clear evidence that chrysotile fibres can cause a fibrogenic and carcinogenic hazard to humans even though the mechanisms by which chrysotile and other fibres cause fibrogenic and carcinogenic effects are not completely understood. The data however, are not adequate for providing quantitative estimates of the risk to humans. This is due to inadequate exposure-response data from inhalation studies, and there are uncertainties concerning the sensitivities of the animal studies predicting human risk (IPCS, 1998).

Carcinogenic effects have not been reported in several oral carcinogenicity studies (IPCS, 1998).

2.5 Effects on humans

Chrysotile can cause asbestosis, lung cancer and mesothelioma in a dose-dependent manner (IPCS, 1998). In most groups of exposed workers, lung cancer is the predominant cause of death related to chrysotile exposure (NICNAS, 1999). Chrysotile is unequivocally a human carcinogen, however the risk to the public associated with its continued use is dependent on the nature of the material to which the public is exposed and the level, frequency and duration of exposure.

2.5.1 Asbestosis

Asbestosis was the first asbestos-related lung disease to be recognised. It is defined as diffuse interstitial fibrosis of the lungs resulting from exposure to asbestos dust. It is this scarring of the lungs which reduces their elasticity and function resulting in breathlessness. It can appear and progress many years after the termination of exposure.

There is some evidence indicating that chrysotile is less potent than amphiboles in causing asbestosis (Wagner et al., 1988; Becklake, 1991). There is also evidence to suggest that fibre size may influence the degree of hazard (NICNAS, 1999).

Studies of workers exposed to chrysotile in different sectors have broadly demonstrated exposure-response or exposure-effect relationships for chrysotile-induced asbestosis, in so far as increasing levels of exposure have produced increases in the incidence and severity of disease. However, there are difficulties in defining this relationship, due to factors such as uncertainties in diagnosis and the possibility of disease progression on cessation of exposure (IPCS, 1998).

In addition, some variation in risk estimates is evident among the available studies. The reasons for the variations are not entirely clear, but may relate to uncertainties in exposure estimates, airborne fibre size distributions in the various industry sectors and statistical models. Asbestotic changes are common following prolonged exposures to 5 to 20 fibres/ml (IPCS, 1998).

2.5.2 Lung cancer

The first reports (Gloyne, 1935; Lynch & Smith, 1935, both cited by IPCS, 1986), suggesting that asbestos might be related to lung cancer occurrence were followed by approximately 60 case reports over the next 20 years. The first epidemiological confirmation of this association was published by Doll (1955, cited by IPCS 1986). Since then, over 30 cohort studies (on various forms of asbestos) have been carried out in industrial populations in several countries. The majority, but not all, have shown an excess lung cancer risk (IPCS 1986).

Combined exposure to asbestos and cigarette smoke synergistically increases the risk of lung cancer (IPCS, 1986). Type of industrial process may affect the incidence of lung cancer, with some studies suggesting the effect is greater for textile workers. The variations may be related to the state and physical treatment of the asbestos in different situations, the dust clouds thus containing asbestos fibres of different physical dimensions (IPCS, 1986).

Exposure to chrysotile is associated with an excess risk of lung cancer. However, for chrysotile the overall relative risks for lung cancer are generally not elevated in the studies of workers in asbestos-cement production and in some of the cohorts of asbestos-cement production workers. The exposure-response relationship between chrysotile and lung cancer risk appears to be 10-30 times higher in studies of textile workers than in studies of workers in mining and milling industries. The relative risks of lung cancer in the textile manufacturing sector in relation to estimated cumulative exposure are, therefore, some 10-30 times greater than those observed in chrysotile mining. The reasons for this variation in risk are not clear, so several hypotheses, including variations in fibre size distribution, have been proposed (IPCS, 1998). Generally, longer chrysotile fibres were used in the textile industry and longer fibres are associated with increased lung tumours (Doll & Peto, 1985 cited by NICNAS, 1999).

The carcinogenic potency of chrysotile compared to the amphiboles has been increasingly debated in the literature. Several authors have concluded that there is sufficient epidemiological evidence to show that chrysotile, at comparable exposures, is less potent than amphiboles in the induction of lung cancer. However, others argue that variations in risk are more related to industry type rather than fibre type and that there is little evidence to indicate a lower risk of lung cancer from exposure to chrysotile (Nicholson and Landrigan, 1994; Stayner et al, 1996 both cited by NICNAS, 1999).

2.5.3 Mesothelioma

Pulmonary mesothelioma is a primary malignant tumour of the mesothelial surfaces, generally affecting the pleura and less commonly the peritoneum. Mesothelioma has been associated with occupational exposure to various types and mixtures of asbestos (including talc containing asbestos), although occupational exposures have not been identified in all cases. The long latency (generally between 35 and 40 years) required for mesothelioma to develop after asbestos exposure has been documented in a number of publications. An increasing proportion of cases have been seen with increasing duration of exposure (IARC, 1987).

Cohort studies of populations of workers using only or predominantly chrysotile-containing products in applications such as construction have not been identified. However, some relevant information is available from population-based analyses of primarily mesothelioma in application workers exposed generally to mixed fibre types (IPCS, 1998). Estimation of the risk of mesothelioma is complicated in epidemiological studies by factors such as the rarity of the disease, the lack of mortality rates in the populations used as reference, and problems in diagnosis and reporting. There is uncertainty regarding risk estimates to chrysotile. Risk estimates used in the calculations were derived from past exposures to relatively high levels of chrysotile (NICNAS, 1999). Current levels of exposure are much lower than the levels estimated in the cohort studies presented in the NICNAS report and as such risk extrapolations may be an overestimate.

Some investigations suggest that the capacity to cause mesothelioma is substantially less for chrysotile than for amphiboles (especially crocidolite) (IPCS, 1986). In contrast, others have concluded that chrysotile is a major cause of mesothelioma in humans and has a similar potency to amphiboles (Smith & Wright, 1996; Huncharek, 1994, both cited by NICNAS, 1999). The US EPA (US EPA, 1989, cited by NICNAS, 1999), in its quantitative assessment of mesothelioma risk, concluded that epidemiological and animal evidence did not conclusively establish differences in mesothelioma hazard for the various asbestos fibre types and as such all asbestos fibres should be regarded as exhibiting similar carcinogenic potency.

There is evidence that fibrous tremolite causes mesothelioma in humans. Since commercial chrysotile may contain fibrous tremolite, it has been hypothesized that the latter may contribute to the induction of mesothelioma in some populations exposed primarily to chrysotile. The extent to which the observed excesses of mesothelioma might be attributed to the fibrous tremolite content has not been resolved (IPCS, 1998). However, mesotheliomas have been reported in animal studies from exposure to uncontaminated experimental chrysotile (e.g. UICC – chrysotile B) (Wagner et al, 1974 cited by NICNAS, 1999; IPCS, 1998). Furthermore, Begin et al. (1992, cited by NICNAS, 1999) reported that in Quebec, mesothelioma rates are as high in the 'Asbestos region' as the 'Thetford mines region', despite much lower tremolite contamination of chrysotile in the former region.

2.5.4 Other malignant diseases

The epidemiological evidence that chrysotile exposure is associated with an increased risk for cancer sites other than the lung or pleura is inconclusive. There is limited information on this issue for chrysotile per se, although there is some inconsistent evidence for an association between asbestos exposure (all forms) and laryngeal, kidney and gastrointestinal tract cancers. A significant excess of stomach cancer has been observed in a study of Quebec chrysotile miners and millers, but possible confounding by diet, infections or other risk factors has not been addressed (IPCS, 1998). In predominantly "chrysotile"-exposed cohorts of workers, there is no consistent evidence of excess mortality from stomach or colorectal cancer.

2.6 Summary of mammalian toxicity and overall evaluation

Fibrosis in many animal species and bronchial and pleural carcinomas in the rat, have been observed following inhalation of chrysotile. In these studies there were no consistent increases in tumour incidence at other sites, and there is no convincing evidence that ingested asbestos is carcinogenic in animals (IPCS, 1986).

Epidemiological studies, mainly on occupational groups, have established that all types of asbestos fibres are associated with diffuse pulmonary fibrosis (asbestosis), bronchial carcinoma (lung cancer), and primary malignant tumours of the pleura and peritoneum (mesothelioma). That asbestos causes cancers at other sites is less well established. Cigarette smoking increases the asbestosis mortality and the risk of lung cancer in persons exposed to asbestos but not the risk of mesothelioma (IPCS, 1986).

3 Human exposure/Risk evaluation

3.1 Food

The extent of asbestos contamination of solid foodstuffs has not been well studied. Asbestos fibres have been detected in beverages. Up to 12 x 106 fibres/litre have been found in soft drinks (IPCS, 1986).

3.2 Air

At remote rural locations, fibre levels (> 5 µm) are generally < 1 fibre/litre (< 0.001 fibre/ml) and in urban air they range from < 1 to 10 fibres/litre (0.001 to 0.01 fibres/ml) or occasionally higher. Airborne levels in residential areas in the vicinity of industrial sources have been found to be within the range of those in urban areas or occasionally slightly higher. Non-occupational indoor levels are generally within the range found in ambient air. The major fibre type observed in the general environment is chrysotile (IPCS, 1986; 1998).

3.3 Water

Available data on effects of exposure to chrysotile asbestos (specifically) in general environment are restricted to those in populations exposed to relatively high concentrations of chrysotile asbestos in drinking-water, particularly from serpentine deposits or asbestos-cement pipe. These include ecological studies of populations in Connecticut, Florida, California, Utah and Quebec, and a case-control study in Puget Sound, Washington, USA (IPCS, 1998). On the basis of these studies, it was concluded that there was little convincing evidence of an association between asbestos in public water supplies and cancer induction. More recent identified studies do not contribute additionally to our understanding of health risks associated with exposure to chrysotile in drinking water (IPCS, 1998).

3.4 Occupational exposure

Workplace concentrations were very high when monitoring first began (in the 1930s). In countries where controls were implemented, the levels generally reduced considerably with time and continue to decline (IPCS, 1998). In contrast, there is less difference between the early results of measurements in both outdoor and indoor non-occupational environments (1970s) and recent data. Based on data mainly from North America, Europe and Japan, in most production sectors workplace exposures in the early 1930s were very high. Levels dropped considerably to the late 1970s and have declined substantially to present day values. In the mining and milling industry in Quebec, the average fibre concentrations in air often exceeded 20 fibres/ml (f/ml) in the 1970s, while they are now generally well below 1 f/ml.

The current main activities resulting in potential chrysotile exposure are: (a) mining and milling; (b) processing into products (friction materials, cement pipes and sheet gaskets and seals, paper and textiles' (c) construction, repair and demolition; (d) transportation and disposal. The asbestos-cement industry is by far the largest user of chrysotile fibres, accounting for about 85% for all use (IPCS, 1998).

Fibres are released during processing, installation and disposal of asbestos-containing products, as well as through normal wear of products in some instances (IPCS, 1998). Manipulation of friable products may be an important source of chrysotile emission.

The conclusions and recommendations of the IPCS 1998 evaluation of chrysotile are that:

Exposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose-dependent manner. No threshold has been identified for carcinogenic risks.

Where safer substitute materials for chrysotile are available, they should be considered for use.

Some asbestos-containing products pose particular concern and chrysotile use in these circumstances is not recommended. These uses include friable products with high exposure potential. Construction materials are of particular concern for several reasons. The construction industry workforce is large and measures to control asbestos are difficult to institute. In-place building materials may also pose risk to those carrying out alterations, maintenance and demolition. Minerals in place have the potential to deteriorate and create exposures.

Control measures, including engineering controls and work practices, should be used in circumstances where occupational exposure to chrysotile can occur. Data from industries where control technologies have been applied demonstrate the feasibility of controlling exposure to levels generally below 0.5 fibres/ml. Personal protective equipment can further reduce individual exposure where engineering controls and work practices prove insufficient.

Asbestos exposure and cigarette smoking have been shown to interact to increase greatly the risk of lung cancer. Those who have been exposed to asbestos can substantially reduce their lung cancer risk by avoiding smoking.

The European Community notification noted that exposure of workers and other users of asbestos-containing products is in general technically extremely difficult to control in practice, and may greatly exceed current limit values on an intermittent basis. It was recognized that a controlled and safe occupational use of chrysotile asbestos could not be established for several working situations like e.g. building sites, repairs, or waste removal. For instance, working under conditions of 0.25 fibres/ml (at the level of the exposure limit value) was still associated with a 35 yr working-life chrysotile-associated cancer risk of 0.77% (0.63% of lung cancers and 0.14% of mesothelioma chrysotile-induced, respectively) when relating to the studies of Doll and Peto (1985). As chrysotile asbestos was widely used and no safe concentration threshold could be established it was decided to severely restrict the use of this asbestos form.

The Chile notification noted that in general the highest exposures to asbestos are amongst the working population whether during manufacture of materials containing asbestos or during installation or demolition. In Chile this means in particular those workers who have been exposed to fibres from the manufacture of construction materials. In the case of brake linings or parts that contain chrysotile asbestos, not only the workers who handle chrysotile during manufacture are exposed to high risk, so are brake repair workshop mechanics who blow off the dust produced by wear. Health controls over this activity are very difficult to implement because of its very nature. In many cases, the workshops involved are small ones that do not have the occupational health means to control the risks.

The Australian risk assessment indicated that exposure to workers is most likely to occur during the handling of raw chrysotile during manufacture, processing and removal of friction products and gaskets. Air monitoring data were analysed from various sources including, Australian industry, the automotive aftermarket survey (carried out by NICNAS), air monitoring in service garages in Western Australia and international exposure data in garage workshops and industries involved in the removal and replacement of chrysotile asbestos friction products and gaskets. Results from these studies indicate that over the past 10 years, samples were less than 1 f/mL (NOHSC national exposure standard for chrysotile at the time of the assessment). Air monitoring data at an Australian friction product manufacturer for the period between 1992 and 1997 indicated that 80% of 461 personal samples (all fibres) were under 0.1 f/ml and two samples were over 0.5 f/ml. Analysis of air monitoring data (between 1991 to 1996) at a manufacturer of compressed chrysotile asbestos fibre sheets showed that approximately 60% of personal samples (all fibres) were less than 0.1 f/ml and one sample was greater than 0.5 f/ml. The NICNAS automotive aftermarket survey indicated that exposure to chrysotile was highest during grinding of brake shoes and cutting of brake linings. The highest personal monitoring result obtained was 0.16 f/ml, during cutting of brake shoes. In view of the health hazards NOHSC has revised the exposure standard for chrysotile to 0.1 f/ml (TWA).

3.5 Para-occupational exposure

Members of the families of chrysotile asbestos workers handling contaminated work clothes, and, in some cases, members of the general population may be exposed to elevated concentrations of building materials for domestic application (e.g. asbestos-cement products and floor tiles), and elevated airborne levels have been measured during the manipulation of these materials (e.g. home construction and renovation by the home owner) (IPCS, 1986).

The Chile notification notes that asbestos fibres are not easily released from asbestos in a cement matrix, in sheeting used in construction. However, people who cut or trim such sheeting using high-speed tools (circular saws or sanders) are exposed to risk from the asbestos-fibre dust given off.

3.6 Public exposure

Based on surveys conducted before 1986, fibre concentrations (fibres $> 5~\mu m$ in length) in outdoor air, measured in Austria, Canada, Germany, South Africa and the USA, ranged between 0.0001 and about 0.01 f/ml, levels in most samples being less than 0.001 f/ml (IPCS, 1998). Fibre concentrations in public buildings, even those with friable asbestos-containing materials, are within the range of those measured in ambient air.

Fibres are released during processing, installation and disposal of chrysotile asbestos-containing materials. In studies reviewed, increases in lung cancer were not observed in four limited ecological epidemiological studies of populations in the vicinity of natural or anthropogenic sources of chrysotile (including the chrysotile mines and mills in Quebec) (IPCS, 1986). Data on incidence or mortality of disease in household contacts of chrysotile workers or in populations exposed to airborne chrysotile in the vicinity of point sources reported since EHC 53 was published in 1986 have not been identified. More recent studies of populations exposed to chrysotile in drinking-water have likewise not been identified (IPCS, 1998).

In general, as exposures experienced by the public will normally be considerably lower and less frequent than those experienced in the industrial environment, the expected lung cancer incidence in the public due to exposure to chrysotile will be lower than those estimated for workers.

The International Programme on Chemical Safety (IPCS) in assessing the risk to the public from asbestos exposure concluded that 'the risks of mesothelioma and lung cancer cannot be quantified and are probably undetectably low' and that 'the risk of asbestosis is virtually zero' (IPCS, 1986).

The Australian risk assessment reported that automotive applications are likely to be the major source of public exposure to chrysotile dusts. A proportion of the end-use products containing chrysotile may be sold directly to the public, particularly automotive friction products and gaskets. In the home mechanic situation, little if any personal protective equipment is likely to be worn when replacing brake pads and shoes, clutch plates or engine gaskets. In the case of gaskets, generation of significant quantities of dust is less likely as the chrysotile is bound into the matrix of the gasket. Similarly dusts created from clutch facings tend to be enclosed in the transmission of the vehicle and most replacement clutch facings do not contain chrysotile. During the changing of brake pads and drum shoes, however, significant exposure is possible. In commercial operations compressed air is generally no longer used to remove excess dust and improved housekeeping practices has reduced exposure levels occupationally and as a consequence, has reduced the likelihood of public exposure from this source. The home mechanic however, may have significant intermittent exposure during the changing of brake pads and shoes (NICNAS, 1999).

Generation of chrysotile dusts at busy traffic intersections, by braking vehicles, is a known source of public exposure. Studies (Jaffrey, 1990 cited by NICNAS, 1999) on the levels of chrysotile fibres at two busy (approximately 2000 vehicles/hr) London intersections found low asbestos levels of between $5.5 \times 10-4$ to $6.2 \times 10-3$ f/mL.

See also information in "occupational" and "para-occupational" sections above.

4 Environmental fate and effects

Serpentine outcroppings occur world-wide. Mineral components, including chrysotile, are eroded through crustal processes and are transported to become a component of the water cycle, sediment population and soil profile. Chrysotile presence and concentrations have been measured in water, air and other units of the crust. Both natural and human activities contribute to fibre aerosolization and distribution (IPCS, 1998).

Chrysotile and its associated serpentine minerals chemically degrade at the surface. This produces profound changes in soil pH and introduces a variety of trace metals into the environment. This has in turn produced measurable effects on plant growth, soil biota (including microbes and insects), fish and invertebrates. Some data indicate that grazing animals (sheep and cattle) undergo changes in blood chemistry following ingestion of grasses grown on serpentine outcrops.

The majority of waste chrysotile from manufacturing is expected to be disposed of to landfill. It can reasonably be expected that chrysotile fibres from end use will reach aquatic systems arising from dust generated during brake wear and to a lesser extent, from disposal to unsecured landfill. Chrysotile is not expected to degrade in aquatic systems although some degradation may occur under acidic conditions (NICNAS, 1999).

There is a paucity of data available as to the effects of chrysotile asbestos in the environment. Data are insufficient to determine if chrysotile asbestos poses any acute or chronic toxicity hazard to plants, birds or land animals (NICNAS, 1999).

5 Environmental Exposure/Risk Evaluation

Environmental effects are not relevant to the risk evaluation used to support the regulatory decisions.

Annex 2 – Details on final regulatory actions reported – Chrysotile

Country Name: Australia

1 Effective date(s) of entry into force of actions

The National Occupational Health and Safety Commission (NOHSC) declared, under Section 38 of the *National Occupational Health and Safety Commission Act 1985*, an amendment to Schedule 2 of the *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC: 1005 (1994)] and *National Model Regulations for the Control of Scheduled Carcinogenic Substances* [NOHSC:1011 (1995)] to prohibit the use of chrysotile, actinolite, anthophyllite, and tremolite asbestos. The declaration was gazetted on 18 June 2003. The Regulations commenced on 31 December 2003.

Reference to the regulatory document

<u>Commonwealth</u> – National Occupational Health and Safety Commission Act 1985; Occupational Health and Safety (Commonwealth Employment) (National Standards) Amendment Regulations 2003 (No. 1) 2003 No. 286 under Occupational Health and Safety (Commonwealth Employment) Act 1991.

<u>Australian Capital Territory – Dangerous Substances (General) Regulation 2004 under Dangerous Substances Act 2004.</u>

<u>New South Wales</u> – Occupational Health and Safety Amendment (Chrysotile Asbestos) Regulation 2003 under the Occupational Health and Safety Act 2000.

<u>Northern Territory</u> – Work Health (Occupational Health and Safety) Regulations under Work Health Act.

<u>Queensland</u> – Workplace Health and Safety Amendment Regulation (No. 4) 2003 under Workplace Health and Safety Act 1995.

<u>South Australia</u> – Occupational Health, Safety and Welfare Regulations 1995 & Health, Safety and Welfare (Asbestos) Variation Regulations 2004 under Occupational Health, Safety and Welfare Act 1986.

<u>Tasmania</u> – Workplace Health and Safety Regulations 1998 under Workplace Health and Safety Act 1995.

<u>Victoria</u> – Occupational Health and Safety (Asbestos) Regulations 2003 under Occupational Health and Safety Act 1985.

<u>Western Australia</u> – Occupational Safety and Health Regulations 1996 under Occupational Safety and Health Act 1984.

<u>Customs</u> – Customs (Prohibited Imports) Amendment Regulations 2003 (no. 10) 2003 no. 321

2 Succinct details of the final regulatory action(s)

All new uses of chrysotile asbestos and goods containing chrysotile asbestos are banned in Australia from 31 December 2003, including the replacement of chrysotile asbestos products when replacement is necessary. It is illegal under the laws of each state and territory to store, sell, install or use any products containing chrysotile asbestos. There are a few exemptions to the ban but these are restricted in scope and operate for a limited time. These are:

- in the use of chrysotile fibre gaskets with saturated or superheated steam, or with substances, classified as dangerous goods. Where compressed asbestos fibre gaskets are to be used with chlorine, the exemption applies for plants used in liquid chlorine service with design process conditions of –45 degrees Celsius and 1500 kPa pressure. *Exemption until 31 December 2004 and, for use with chlorine until 31 December 2006*.
- products consisting of a mixture of asbestos with a phenol formaldehyde resin or with a cresylic formaldehyde resin used in vanes for rotary vacuum pumps or rotary compressors; or split face seals of at least 150 mm in diameter used to prevent leakage of water from cooling water pumps in fossil fuel electricity generating stations. Exemption until 31 December 2007.
- diaphragms for use in electrolytic cells in existing electrolysis plants for chlor-alkali manufacture. *Exemption until 31 December 2006.*

for the Australian Defence Organisation (ADO) to use chrysotile parts and components which the ADO considers to be mission-critical, and where there is no known suitable, non-chrysotile alternative. This exemption will be regulated in detail by the Safety Rehabilitation Compensation Commission. *Exemption until* 31 December 2007.

3 Reasons for action

Human Health

To remove almost all human exposure to chrysotile asbestos and thereby minimise the risks to the health of workers and consumers.

4 Basis for inclusion into Annex III

4.1

Risk evaluation

A sixt assumed a Catalog of the same transfer of the Cart assumed to the Cart assumed to the Cart assumed to the catalog of th

A risk assessment of chrysotile was undertaken and the final report was published in February 1999. It assessed the occupational, public health and environmental risks associated with uses and applications of chrysotile in Australian industry. It also assessed the feasibility of substitution of chrysotile materials and voluntary and/or legislative action for reducing potential health and safety risks arising from manufacture and import of chrysotile products. The risk assessment concluded that human exposure to chrysotile is associated with an excess risk of asbestosis, lung cancer and mesothelioma.

4.2 Criteria used

Regulatory actions were taken on the basis of unacceptable risk to human health. The risk assessment into chrysotile asbestos concluded that it causes asbestosis, lung cancer and mesothelioma in humans and animals in a dose related manner. The Australia Mesothelioma Program reports that Australia has the highest incidence of mesothelioma in the world. It has been estimated that the lifetime risk of lung cancer, based on the best available epidemiological data is up to 173 additional cancers per 100,000 workers exposed to a daily average of 1 chrysotile fibre/mL. Extrapolation for lower exposures provides lifetime risk estimates (per 100,000 population) of 86 and 17 for exposure to 0.5 and 0.1 f/mL, respectively (NOHSC, 1995 cited by NICNAS, 1999).

Relevance to other States and Region

Similar exposure scenarios to chrysotile as in Australia may be found in other countries. By removing the exposure to chrysotile asbestos it may be possible to reduce future cases of asbestosis, lung cancer and mesothelioma in workers and public.

5 Alternatives

The risk assessment assessed the feasibility of the substitution of chrysotile. It found that alternatives have been developed for most uses of chrysotile in Australia. Chrysotile has been replaced for example in railway blocks, cement sheeting, tubes and piping, roofing tiles, textiles, fibre insulation and brake disc pads. International research into alternatives for asbestos friction products has led to the development of a number of alternative materials that are claimed to exhibit equal or higher performance standards to chrysotile (NICNAS, 1999).

6 Waste management

The risk assessment concluded that disposal of used chrysotile asbestos parts to standard municipal landfills is acceptable. However, it was recommended that all workplace asbestos waste be collected and disposed of by licensed hazardous waste contractors.

7 Other

Chrysotile is listed in the National Occupational Health and Safety Commission's (NOHSC) Hazardous Substances Information System (HSIS) with the classification: Carcinogen in Category 1: may cause cancer by inhalation (Carc. Cat. 1; R49) Toxic: danger of serious damage to health by prolonged exposure through inhalation (T; R48/23).

NOHSC has revised the exposure standard for chrysotile to 0.1 f/ml (TWA) from 1 f/ml (TWA).

Country Name: Chile

1 Effective date(s) of entry into force of actions
Reference to the regulatory document

Supreme Decree No. 656 entered into force 180 days after its publication in the Official Journal, on 12 July 2001.

Supreme Decree No. 656 of 12 September 2000, Official Journal, 13 January 2001

2 Succinct details of the final regulatory action(s)

Production, importation, distribution, sale and use of crocidolite and any material or product containing it are prohibited.

Production, importation, distribution, sale and use of construction materials containing any type of asbestos are prohibited.

Production, importation, distribution, sale and use of chrysotile, actinolite, amosite, anthophyllite, tremolite and any other type of asbestos, or mixture thereof, for any item, component or product that does not constitute a construction material are prohibited, with certain specific exceptions.

3 Reasons for action

Human Health

To reduce exposure to asbestos amongst the working population during manufacture of material containing asbestos or during installation or demolition.

4 Basis for inclusion into Annex III _

Annex III
4.1 Risk evaluation

The foreign literature and analysis of domestic cases of asbestosis and mesothelioma indicate that those at greatest risk are workers who handle asbestos fibres for various uses.

In Chile, this means in particular those workers who have been exposed to fibres from the manufacture of construction materials.

No epidemiological precedents are known that show that there is a risk to the population from asbestos which is already included within a cement matrix in sheeting used in construction, given that the asbestos fibres are not easily released from the matrix. Nor is there any significant known risk from consuming water piped through asbestos cement piping.

Nevertheless, people who cut or trim such sheeting using high-speed tools (circular saws or sanders) are exposed to risk from asbestos-fibre containing dust given off.

In the case of brake lining or parts that contain asbestos, not only the workers who handle asbestos during manufacture are exposed to high risk, so are brake repair workshop mechanics who blow off the dust produced by wear. It should be noted that health controls over this activity are very difficult to implement because of its very nature. In many cases, the workshops involved are small ones that do not have the occupational health means to control the risks.

4.2 Criteria used

Unacceptable risk to workers.

All types of asbestos are hazardous to health to varying degrees depending on the form of exposure (it has been shown that the risk is from inhalation), the class of asbestos (blue asbestos is the most toxic), the size of the fibres, fibre concentration and interaction with other factors (tobacco smoking potentiates the effects). Generally speaking, the highest exposures are amongst the working population whether during manufacture of the materials containing asbestos or during installation or demolition.

Relevance to other States and Region

The regulatory action prohibits imports of asbestos in general, whatever the country of origin. Therefore no country may export asbestos to Chile except in specific cases, which exclude material and inputs for construction material and must be expressively authorized by Health Authority.

5 Alternatives

It has been proved that it is feasible to replace asbestos with other fibres in manufacturing fibre-cement materials and still obtain products of similar quality. In fact, the company producing the greatest quantity of panels and sheeting for dwellings in Chile has replaced asbestos with other fibres such as cellulose.

In case of brake parts, asbestos-containing and asbestos-free brake pads and linings are in use until the existing in use asbestos-containing brake pads and linings at the time of publication of the prohibition should be replaced.

- 6 Waste management
- 7 Other

Chrysotile is listed in the Chilean Regulations on Basic Sanitary and Environmental Conditions in Workplaces (Supreme Decree No. 594), with the classification: A.1 Proved Human Carcinogen.

In accordance with the Chilean Regulations on Basic Sanitary and Environmental Conditions in Workplaces (Supreme Decree No. 594), chrysotile fibres exposure limit value for workers is 1.6 fibres/cc determined by means of a contrast microscope with magnifying potency of 400–450, in a sample from a membrane filter, counting fibres greater than 5 μm length and a ratio length to diameter equal to or greater than 3:1.

Country Name: European Community

1 Effective date(s) of entry into force of actions Regulatory action was first taken in 1983, in relation to crocidolite. Subsequently, such action has progressively been extended to all forms of asbestos. The latest regulatory action entered in force on 26.8.1999 (OJ L 207 of 6.8 1999, p. 18). Member States of the E.C. were obliged to implement the necessary national legislation at the latest by 1st January 2005.

Reference to the regulatory document

Directive 1999/77/ E.C. of 26.7.1999 (Official Journal of the European Communities (OJ) L207 of 6.8.99, p.18) adapting to technical progress for the sixth time Annex 1 to Directive 76/769/EEC of 27.7.1976 (OJ L 262 of 27.9.1976, p.24). Other relevant Regulatory Actions: Directives 83/478/EEC of 19.91983 (OJ L 263 of 24.9.1983, p.33), 85/610/EEC of 20.12.1985 (OJ L 375 of 31.12.1985, p.1), 91/659/EEC of 3.12.1991 (OJ L 363 of 31.12.91, p.36)

2 Succinct details of the final regulatory action(s)

The placing on the market and use of chrysotile fibres and products containing these fibres added intentionally are prohibited.

The placing on the market and use of chrysotile may be allowed by Member States for diaphragms for existing electrolysis installations until they reach the end of their service life, or until suitable asbestos-free substitutes become available, whichever is the sooner. The derogation will be reviewed before 1 January 2008.

The use of products containing asbestos fibres that were already installed and/or in service before the implementation date of Directive 1999/77/ E.C. by the Member State concerned could continue to be authorised until they are disposed of, or reach the end of their service life. However, Member States could, for reasons of protection of health, prohibit within their territory the use of such products before they are disposed of or reach the end of their service life.

- 3 Reasons for action
- Prevent health effects (asbestosis, lung cancer, mesothelioma) for workers and general public.
- 4 Basis for inclusion into Annex III
- 4.1 Risk evaluation

A comparison of asbestos with possible substitutes by the Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) concluded that all forms of asbestos are carcinogenic to humans and are likely to present a greater risk than substitutes (CSTEE, 1998).

4.2 Criteria used
Relevance to other
States and Region

Standard E.C. criteria used for evaluation of exposure.

Health problems similar to the ones experienced in the E.C. may occur in states where the substance is used in industrial plants and/or as building material, especially in developing countries, where the use of asbestos is still growing. A ban protects health of workers and of the general public.

5 Alternatives

The risk assessment under taken by the CSTEE on chrysotile asbestos and candidate substitutes concludes that, both for the induction of lung and pleural cancer and lung fibrosis and for other effects, it is unlikely that the alternatives cellulose fibres, PVA fibres or P-aramid fibres pose an equal or greater risk than chrysotile asbestos. With regard to carcinogenesis and induction of lung fibrosis the risk is regarded to be lower (CSTEE, 1998).

6 Waste management

In accordance with Council Directive 87/217/EEC (OJ L 85, 28.3.1987, p.40), as amended by Council Directive 91/692/EEC (OJ L 377, 31.12.1991, p.48) on the demolition of buildings, structures and installations containing asbestos and the removal of asbestos or materials containing asbestos therefrom or materials containing asbestos involving the release of asbestos fibres or dust must not cause significant environmental pollution.

Construction materials have been classified as hazardous waste and will thus, as from 1 January 2002, have to be disposed of in line with the obligations laid down in Council Directive 91/689/EEC (OJ L 377, 31.12.1991, p.20). In addition, the Commission is considering measures to promote the practice of selective demolition in order to segregate the hazardous waste present in construction materials and ensure their safe disposal.

7 Other

In accordance with Council Directive 83/477/EEC (OJ L 263, 24.9.1983, p.25), as amended by Council Directive 91/382/EEC (OJ L 206, 29.7.1991, p.16) the European Community exposure limit values for workers are currently 0.6 fibres/ml for chrysotile. Exposure limit values for workers: Proposal still under consideration before the Council and the European Parliament: in 2001 the European Commission proposed (OJ C 304 E 30/10/2001, p.175) that these limits be replaced by a reduced, single limit value of 0.1 fibres/ml for all forms of asbestos.

Annex 3 – Addresses of designated national authorities

AUSTRALIA		
C Assistant Secretary Australian Government of the Department of the Environment & Heritage John Gorton Building	Phone Fax Telex e-mail	+61 2 6274 1622 +61 2 6274 1164 mark.hyman@deh.gov.au
King Edward Terrace		
Parkes ACT 2600 Australia Mr Mark Hyman		

CHILE		
C	Phone	+56 2 6300575/6300625
Department of Environmental Health	Fax	+56 2 664 9150
Ministry of Health	Telex	
Mac Iver 459 Piso 8	e-mail	pchavez@minsal.gov.cl
Santiago		
Chile		
Ms Paulina Chavez		

EUROPEAN COMMUNITY		
СР	Phone	+32 2 2994860
DG Environment	Fax	+32 2 2967617
European Commission	Telex	
Rue de la Loi 200	e-mail	Klaus.berend@cec.eu.int
B-1049 Brussels		
Belgium		
Klaus Berend		

C Industrial chemicals

CP Pesticides, industrial chemicals

P Pesticides

Annex 4 – References – Chrysotile

Regulatory action

Australia

National Occupational Health and Safety Commission Act 1985; Occupational Health and Safety (Commonwealth Employment) (National Standards) Amendment Regulations 2003 (No. 1) 2003 No. 286 under Occupational Health and Safety (Commonwealth Employment) Act 1991; Australian Capital Territory — Dangerous Substances (General) Regulation 2004 under Dangerous Substances Act 2004; New South Wales — Occupational Health and Safety Amendment (Chrysotile Asbestos) Regulation 2003 under the Occupational Health and Safety Act 2000; Northern Territory — Work Health (Occupational Health and Safety) Regulations under Work Health Act; Queensland — Workplace Health and Safety Amendment Regulation (No. 4) 2003 under Workplace Health and Safety Act 1995; South Australia — Occupational Health, Safety and Welfare Regulations 1995 & Health, Safety and Welfare (Asbestos) Variation Regulations 2004 under Occupational Health, Safety and Welfare Act 1986; Tasmania — Workplace Health and Safety Regulations 1998 under Workplace Health and Safety Act 1995; Victoria — Occupational Health and Safety (Asbestos) Regulations 2003 under Occupational Health and Safety Act 1985; Western Australia — Occupational Safety and Health Regulations 1996 under Occupational Safety and Health Act 1984; Customs — Customs (Prohibited Imports) Amendment Regulations 2003 (no. 10) 2003 no. 321.

Chile

Supreme Decree No. 656 of 12 September 2000, Official Journal, 13 January 2001

European Community

Directive 1999/77/ E.C. of 26.7.1999 (Official Journal of the European Communities (OJ) L207 of 6.8.99, p.18) adapting to technical progress for the sixth time Annex 1 to Directive 76/769/EEC of 27.7.1976 (OJ L 262 of 27.9.1976, p.24). Other relevant Regulatory Actions: Directives 83/478/EEC of 19.91983 (OJ L 263 of 24.9.1983, p.33), 85/610/EEC of 20.12.1985 (OJ L 375 of 31.12.1985, p.1), 91/659/EEC of 3.12.1991 (OJ L 363 of 31.12.91, p.36)

Other Documents

Becklake MR (1991). The epidemiology of asbestosis. In: D. Liddell and K. Miller (eds) Mineral fibres and health, Florida, CRC Press Boca Raton.

Begin R, Masse S, Rola-Pleszczynski M, Boctor M & Drapeau G (1987) Asbestos exposure dose – bronchoalveolar milieu response in asbestos workers and the sheep model: evidences of a threshold for chrysotile-induced fibrosis. In: Fisher GL & Gallo MA ed. Asbestos toxicity. New York, Basel, Marcel Dekker Inc., pp 87-107.

Bissonnette E, Dubois C, & Rola-Pleszczynski M (1989) Changes in lymphocyte function and lung histology during the development of asbestosis and silicosis in the mouse. Res Commun Chem Pathol Pharmacol, 65: 211-227.

Bunn W B, Bender JR, Hesterberg TW, Chase G R, & Konzen J L (1993) Recent studies of man-made vitreous fibers: Chronic animals inhalation studies. J Occup Med, 35: 101-113.

Coffin D L, Cook P M & Creason J P (1992) Relative mesothelioma induction in rats by mineral fibres: comparison with residual pulmonary mineral fibre number and epidemiology. Inhal Toxicol, 4: 273-300

CSTEE (1998) Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) – Opinion on Chrysotile asbestos and candidate substitutes expressed at the 5th CSTEE plenary meeting, Brussels, 15 September 1998 http://europa.eu.int/comm/food/fs/sc/sct/out17 en.html

Davis J M G, Addison J, Bolton R E, Donaldson K, & Jones A D. (1986) Inhalation and injection studies in rats using dust samples from chrysotile asbestos prepared by a wet dispersion method. Br J Path 67: 113-129.

Davis J M G, Bolton R E, Douglas A N, Jones AD, & Smith T (1998) The effects of electrostatic charge on the pathogenicity of chrysotile asbestos. Br J Ind Med, 45: 337-345.

Directive 1999/77/ E.C. of 26.7.1999 (Official Journal of the European Communities (OJ) L207 of 6.8.99, p.18) adapting to technical progress for the sixth time Annex I to Directive 76/769/EEC of 27.7.1976 (OJ L 262 of 27.9.1976, p. 24).

Directive 2001/59/ E.C. of 6.8.2001 (Official Journal of the European Communities (OJ)) L225/1.

Doll R (1955) Mortality from lung cancer in asbestos workers. British Journal of Industrial Medicine 12: 81-86.

Doll R & Peto J (1985) Asbestos: Effects on health of exposure to asbestos, Report commissioned by the HSE.

Dunnigan J (1988) Linking chrysotile asbestos with mesothelioma. American Journal of Industrial Medicine 14: 205-209.

E.C. (1997) European Commission DGIII, Environmental Resources Management. Recent assessments of the hazards and risks posed by asbestos and substitute fibres, and recent regulation of fibres worldwide. Oxford.

E.C. (2001) Commission Directive 2001/59/European Community August 2001.

Fasske E (1988) Experimental lung tumors following specific intrabronchial application of chrysotile asbestos. Respiration, 53: 111-127.

Gibbs G W, Valic F, Browne K (1994) Health risks associated with chrysotile asbestos. Annals of Occupational Hygiene 38(4): 399-426.

Gloyne S R (1935) Two cases of squamous carcinoma of the lung occurring in asbestosis. Tuberculosis 17:5.

IARC (1987) IARC monographs on the evaluation of carcinogenic risks to humans: overall evaluations of carcinogenicity: updating of IARC monographs volumes 1 to 42 (supplement 7), International Agency for Research on Cancer, Lyon.

International Labour Organisation (1986) Convention No. 162 and Recommendation 172 concerning safety in the use of asbestos [ILO]. International Labour Office, 1986.

International Standards Organisation (1984) Asbestos reinforced cement products – Guidelines for on-site work practices. ISO 7337. First edition 1984-07-01.

IPCS (1986) Environmental Health Criteria 53: Asbestos and other Natural Mineral Fibres. World Health Organisation, Geneva.

IPCS (1998) Environmental Health Criteria 203: Chrysotile asbestos. World Health Organisation, Geneva.

Le Bouffant L, Daniel H, Henin J P, Martin J C, Normand C, Tichoux G, & Trolard F (1987) Experimental study on long-term effects of inhaled MMMF on the lungs of rats. Ann Occup Hyg, 31:765-790.

Lemaire I (1985) Characterization of the bronchoalveolar cellular response in experimental asbestosis: Different reactions depending on the fibrogenic potential. Am Rev Respir Dis, 131: 144-149.

Lemaire I (1991) Selective differences in macrophage populations and monokine production in resolving pulmonary granuloma and fibrosis. Am J Pathol, 138: 487-495.

Lemaire I, Nadeau D, Dunnigan J, & Masse S (1985) An assessment of the fibrogenic potential of very short 4T30 chrysotile by intratracheal instillation in rats. Environ Res, 36: 314-326.

Lemaire I, Dionne PG, Nadeau D, & Dunnigan J (1989) Rat lung reactivity to natural and man-made fibrous silicates following short-term exposure. Environ Res, 48: 193-210.

Lynch K M and Smith W A (1935) Pulmonary asbestosis. III. Carcinoma of lung in asbestos-silicosis. American Journal of Cancer 24:56.

National primary drinking water regulations—synthetic organic chemicals and inorganic chemicals, final rule, 56 Federal Register 3526 (January 30, 1991).

NICNAS (1999) Chrysotile asbestos: priority existing chemical no. 9: full public report. Sydney, National Industrial Chemicals Notification and Assessment Scheme.

Royal Society of Canada: (1996). A review of the INSERM Report on the health effects of exposure to asbestos: Report of the Expert Panel on Asbestos Risk.

Sebastien P, Begin R, & Masse S (1990) Mass number and size of lung fibres in the pathogenesis of asbestosis in sheep. Int J Exp Pathol, 71: 1-10.

US (2001) U.S National Toxicology Program '9th Report on Carcinogens', revised Jan 2001.

Wagner JC, Berry BG, Hill RJ, Munday DE, & Skidmore JW (1984) Animal experiments with MMM(V)F. Effects of inhalation and intraperitoneal inoculation in rats. In: Proceedings of a WHO/IARC conference: Biological Effects of Man-made Mineral Fibres. WHO, Regional Office for Europe, Copenhagen, 209-233.

Wagner JC, Newhouse ML, Corrin B et al. (1988) Correlation between fibre content of the lung and disease in east London asbestos factory workers. British Journal of Industrial Medicine, 45(5): 305-308.

29